

What is claimed is:

1. A method to inhibit airway hyperresponsiveness in a mammal, comprising administering to a mammal an agent that binds to and activates a calcitonin gene related peptide (CGRP) receptor in the lungs of said mammal, wherein said mammal has, or is at risk of developing, airway hyperresponsiveness.

2. The method of Claim 1, wherein said airway hyperresponsiveness is allergen-induced airway hyperresponsiveness.

3. The method of Claim 2, wherein said mammal has been sensitized to an allergen and has been exposed to, or is at risk of being exposed to, an amount of said allergen that is sufficient to induce airway hyperresponsiveness (AHR) in said mammal in the absence of said agent.

4. The method of Claim 1, wherein said method further comprises monitoring said mammal to detect whether AHR in said mammal is inhibited, wherein if AHR is detected in said mammal, additional amounts of said agent are administered until AHR is not detected in said mammal.

5. The method of Claim 1, wherein said agent is administered within a time period of between 48 hours or less prior to exposure to an AHR provoking stimulus that is sufficient to induce AHR, and within 48 hours or less after the detection of the first symptoms of AHR.

6. The method of Claim 1, wherein said agent is administered upon the detection of the first symptoms of AHR.

7. The method of Claim 1, wherein said agent is administered within 1 hour after the detection of the first symptoms of AHR.

8. The method of Claim 1, wherein said agent is administered within 12 hours or less prior to exposure to a AHR provoking stimulus that is sufficient to induce AHR.

9. The method of Claim 1, wherein said agent is administered within 2 hours or less prior to exposure to a AHR provoking stimulus that is sufficient to induce AHR.

10. The method of Claim 1, wherein said agent is administered to said mammal every one to two days.

11. The method of Claim 1, wherein said agent is selected from the group consisting of CGRP, a fragment of CGRP that binds to and activates a CGRP receptor, and a homologue of CGRP that binds to and activates a CGRP receptor.

12. The method of Claim 1, wherein said agent is administered at a dose of from about  $0.1 \mu\text{g} \times \text{kilogram}^{-1}$  and about  $20 \mu\text{g} \times \text{kilogram}^{-1}$  body weight of said mammal.

13. The method of Claim 1, wherein said agent is administered at a dose of from about  $0.1 \mu\text{g} \times \text{kilogram}^{-1}$  and about  $10 \mu\text{g} \times \text{kilogram}^{-1}$  body weight of said mammal.

14. The method of Claim 1, wherein said agent is administered at a dose of from about  $0.1 \mu\text{g} \times \text{kilogram}^{-1}$  and about  $5 \mu\text{g} \times \text{kilogram}^{-1}$  body weight of said mammal.

15. The method of Claim 1, wherein said agent is a product of rational drug design that binds to and activates a CGRP receptor.

16. The method of Claim 1, wherein said agent is an antibody that selectively binds to and activates said CGRP receptor.

17. The method of Claim 16, wherein said antibody is a divalent antibody.

18. The method of Claim 16, wherein said antibody is a bivalent antibody, wherein said antibody selectively binds to said CGRP receptor and to an antigen on a cell selected from the group consisting of a lung smooth muscle cell and a lung epithelial cell.

19. The method of Claim 1, wherein said agent is an antigen binding fragment of an antibody that selectively binds to and activates said CGRP receptor.

20. The method of Claim 1, wherein said agent is targeted to cells in the lung of said mammal selected from the group consisting of smooth muscle cells and epithelial cells.

21. The method of Claim 1, wherein said agent is administered by direct delivery of said agent to the lung of said mammal.

22. The method of Claim 1, wherein said agent is administered by aerosol delivery.

23. The method of Claim 1, wherein said agent is administered by parenteral delivery.

24. The method of Claim 1, wherein said agent is administered by oral delivery.

25. The method of Claim 1, wherein administration of said agent reduces the airway hyperresponsiveness of said mammal such that the FEV<sub>1</sub> value of said mammal is improved by at least about 5%.

26. The method of Claim 1, wherein administration of said agent prevents airway hyperresponsiveness in said mammal when administered prior to exposure of said mammal to a AHR provoking stimulus that is sufficient to induce AHR.

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27. The method of Claim 1, wherein said agent is administered to said mammal in conjunction with another agent selected from the group consisting of: corticosteroids, (oral, inhaled and injected),  $\beta$ -agonists (long or short acting), leukotriene modifiers (inhibitors or receptor antagonists), antihistamines, phosphodiesterase inhibitors, sodium cromoglycate, nedocrilal, and theophylline.

28. The method of Claim 1, wherein said agent is administered to said mammal in conjunction with a CGRP receptor activity modifying protein (RAMP).

29. The method of Claim 1, wherein said agent is administered in a pharmaceutically acceptable excipient.

30. The method of Claim 1, wherein said mammal is a human.

31. A method to identify an agent for reducing airway hyperresponsiveness in a mammal, comprising:

- a. contacting a calcitonin gene related peptide (CGRP) receptor with a putative regulatory agent;
- b. detecting whether said putative regulatory agent binds to said CGRP receptor;
- c. administering a putative regulatory agent which binds to said CGRP receptor to a non-human test mammal in which airway hyperresponsiveness can be induced and detecting whether the putative regulatory agent reduces airway hyperresponsiveness in said test mammal upon induction of airway hyperresponsiveness in the presence of said putative regulatory agent as compared to in the absence of said putative regulatory agent;

wherein putative regulatory agents that bind to said CGRP receptor and that reduce airway hyperresponsiveness in the test mammal are identified as agents which reduce airway hyperresponsiveness.

32. The method of Claim 31, wherein said step (c) of administering comprises administering said putative regulatory agent which binds to said CGRP receptor to a non-human test mammal that has been sensitized to an allergen and detecting whether the putative regulatory agent reduces airway hyperresponsiveness in said test mammal when said mammal is challenged with said allergen, as compared to in the absence of said putative regulatory agent;

wherein putative regulatory agents that bind to said CGRP receptor and that reduce airway hyperresponsiveness in the test mammal are identified as agents which reduce allergen-induced airway hyperresponsiveness.

33. The method of Claim 31, wherein said CGRP receptor is a soluble receptor.

34. The method of Claim 31, wherein in part (a), said CGRP receptor is expressed by a cell, and wherein said step (b) of detecting further comprises detecting whether said CGRP receptor is activated by said putative regulatory compound.

35. The method of Claim 31, wherein said non-human test mammal is a mouse.
36. The method of Claim 31, wherein said putative regulatory agent is a product of rational drug design.
37. The method of Claim 31, wherein said putative regulatory agent is an antibody.
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